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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte MICHAEL G. ROSENBLUM

Appeal 2009-009632
Application 10/676,725
Technology Center 1600

Decided: February 24, 2010

Before DEMETRA J. MILLS, ERIC GRIMES and STEPHEN WALSH, *Administrative Patent Judges*.

GRIMES, *Administrative Patent Judge*.

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 involving claims to a method of treating cancer, which the Examiner has rejected as obvious. We have jurisdiction under 35 U.S.C. § 6(b). We affirm.

STATEMENT OF THE CASE

"Many tumors or cancer cells express membrane-bound or cytoplasmic antigens or antigenic determinants which are either expressed

very weakly or not at all by normal cells. . . . These abnormally expressed antigens are known as tumor-associated antigens." (Spec. 2.)

The Specification discloses that "[a]ntibodies, coupled to drugs, have been used as a delivery system by which the drug is targeted to a specific tumor cell type against which the antibody is directed" (*id.* at 3). The Specification states that "conjugation of antibodies to biological response modifiers such as tumor necrosis factor and the use of such conjugates as specific delivery system to target tissues or cells has not heretofore been possible" (*id.* at 4).

Claims 7, 10, 13, 14, 16, 21, and 23-32 are on appeal. Claim 26 is representative and reads as follows:

26. A method of treating cancer in a human patient in need of such treatment, the method comprising the steps of:

(a) identifying a patient having a tumor, which tumor comprises cells for targeting and wherein those cells comprise a cell surface antigenic marker at concentrations in excess of that found at other non-target sites;

(b) obtaining a composition comprising a protein with an antigen recognition site directed toward a cell surface associated antigen conjugated or fused to the biological response modifier, wherein it has been determined that cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site; and

(c) administering an amount of the composition to the patient effective to treat the cancer.

The claims stand rejected under 35 U.S.C. § 103(a) as follows:

- Claims 7, 10, 13, 14, 21, 24-29, and 32 based on Scannon[1] and Ferris,[2] with evidence provided by Kirkwood[3] (Ans. 4);
- Claim 16 based on Scannon, Ferris, and Blick[4] (Ans. 7);
- Claim 23 based on Scannon, Ferris, and Ghose[5] (Ans. 8);
- Claims 7, 24, and 26-30 based on Frankel[6] and Ferris (Ans. 10); and
- Claims 7, 24, 26-29, and 31 based on Mattes[7] and Ferris (Ans. 12).

REJECTIONS BASED ON SCANNON AND FERRIS

Issue

The Examiner has rejected claims 7, 10, 13, 14, 21, 24-29, and 32 as obvious based on Scannon and Ferris, with evidence provided by Kirkwood; claim 16 as obvious based on Scannon, Ferris, and Blick; and claim 23 as obvious based on Scannon, Ferris, and Ghose.

The Examiner finds that Scannon discloses "a method of treating melanoma in humans comprising administering an antibody-ricin A toxin conjugate, wherein the antibody of the conjugate binds the melanoma-specific antigen of 240kD" (Ans. 4) but does not teach conjugating the

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[1] Scannon et al., US 4,590,071, May 20, 1986
[2] Ferris et al., 4,771,128, Sept. 13, 1988
[3] John M. Kirkwood et al., "*Scintigraphic Detection of Metastic Melanoma Using Indium 111/DTPA Conjugated Anti-gp240 Antibody (ZME-018),*" 5 JOURNAL OF CLINICAL ONCOLOGY 1247-1255 (1987)
[4] Mark Blick et al., "*Phase I Study of Recombinant Tumor Necrosis Factor in Cancer Patients*," 47 CANCER RESEARCH 2986-2989 (1987)
[5] Tarunendu Ghose et al., "*The Design of Cytotoxic-Agent-Antibody Conjugates*," 3 CRC CRITICAL REVIEWS THERAPEUTIC DRUG CARRIER SYSTEMS 263-359
[6] Frankel et al., US 4,753,894, June 28, 1988
[7] Mattes et al., US 4,666,845, May 19, 1987

antibody to a biological response modifier such as TNF (*id.* at 5). The Examiner cites Kirkwood as evidence that the antigen bound by Scannon's antibody is the same cell-surface antigen that is recognized by antibody XME-018 (Ans. 5). The Examiner finds that Ferris teaches "how to make an antibody conjugated to the biological response modifier TNF" to target tumor cells (*id.* at 5-6). The Examiner concludes that it would have been obvious to "substitute TNF for the ricin A toxin of the antibody conjugate taught by Scannon et al because Ferris et al teach that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody" (*id.* at 6).

Appellant contends that Scannon does not describe its 240 kD melanoma antigen as a cell-surface antigen and the Examiner has not shown that Scannon's and Kirkwood's antigens are the same (Appeal Br. 7-9). Appellant also contends that the claims require "the determination that cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site" (*id.* at 9) and that "[n]owhere does the Answer explain how Scannon teaches the concept of *pre-testing* the patient prior to administering the immunotoxin" (Reply Br. 3).

The issues with respect to the rejections based on Scannon and Ferris are:

Has Appellant shown that the Examiner erred in finding that Scannon discloses an antibody that binds to a cell-surface antigen?

and

Do the claims require confirming that a patient's tumor expresses the specific antigen that is recognized by the agent that is administered?

Findings of Fact

1. Scannon discloses "chemically bonding the A chain of a toxic lectin, such as ricin or abrin, with monoclonal antibodies specific to human melanoma (MoAbHM) to form cytotoxic products called conjugates. . . . [T]he MoAbHM functions as the delivery vehicle for the toxic A chain, delivering the toxin specifically to human melanoma cells." (Scannon, col. 1, ll. 57-65.)

2. Scannon discloses that a preferred conjugate comprises "the XMMME-001 variety of monoclonal antibodies" (*id.* at col. 2, ll. 8-9).

3. Scannon discloses that the XMMME-001 antibody bound to 8 of 10 human melanoma cell lines tested and did not bind to any of the normal cells or non-melanoma cancer cells tested (*id.* at col. 6, ll. 22-50).

4. Ferris discloses that "'[i]mmunoconjugates' may be prepared by covalently linking [monoclonal] antibodies to any of a number of cytotoxic agents" (Ferris, col. 1, ll. 21-23).

5. Ferris discloses that "certain cytokines such as tumor necrosis factor (TNF) are cytotoxic" (*id.* at col. 2, ll. 61-62).

6. In a working example, Ferris describes an immunoconjugate comprising TNF and a monoclonal antibody (*id.* at col. 6, l. 13 to col. 7, l. 13).

7. Kirkwood discloses that "[g]p240 is a melanoma-associated antigen that has exhibited greater restriction to melanoma than other antigens. Gp240 has been found in 80% to 94% of melanoma specimens studied." (Kirkwood 1247).

8. Kirkwood discloses that antibody ZME-018 binds to gp240 (*id.*, abstract).

9. In response to Appellant's argument that Scannon does not disclose a cell-surface antigen, the Examiner cited Ashcroft,[8] Ferrone,[9] Martin,[10] and Oratz[11] (Ans. 15-16).

10. Oratz discloses that the immunoconjugate "XMMME-001-RTA" is "an antimelanoma monoclonal antibody conjugated to ricin A chain" (Oratz 345).

11. Oratz cites Scannon as the source of immunoconjugate XMMME-001-RTA (*id.* at 346 (citing reference 9)).

12. Martin cites Oratz in support of its statement that "[a]ntibodies directed against HMWMAA have been used clinically to target toxic agents to melanomas" (Martin 738).

13. Martin describes HMWMAA as a "melanoma surface antigen" (*id.*).

14. Ferrone discloses that HMW-MAA is a membrane-bound antigen with a certain range of "[d]ensity on tumor cells" (Ferrone 458, Table 1).

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[8] Jared M. Ashcroft et al., "*Fullerene (C_{60}) immunoconjugates: interaction of water-soluble C_{60} derivatives with the murine anti-gp240 melanoma antibody*," CHEM. COMMUN. 3004-3006 (2006)
[9] Soldano Ferrone et al., "*Human High Molecular Weight-melanoma Associated Antigen as a Target for Active Specific Immunotherapy*," 15 Journal of Dermatology 457-465 (1988)
[10] F. Martin et al., "*Retroviral Vector Targeting to Melanoma Cells by Single-Chain Antibody Incorporation in Envelope*," 9 HUMAN GENE THERAPY 737-746 (1998)
[11] R. Oratz et al., "*Antimelanoma Monoclonal Antibody-Ricin A Chain Immunoconjugate (XMMME-001-RTA) Plus Cyclophosphamide in the Treatment of Metastatic Malignant Melanoma: Results of a Phase II Trial*," 9 JOURNAL OF BIOLOGICAL RESPONSE MODIFIERS 345-354 (1990)

15. Ashcroft discloses that gp240, the antigen bound by antibody ZME-018, is "also known as the high molecular weight melanoma-associated antigen, HMWMAA" (Ashcroft 3004).

16. Blick discloses that "activated macrophages produce TNF-α, whereas mitogen-stimulated lymphocytes produce TNF-β. . . . Both cytokines are known to have cytostatic and cytotoxic effects *in vitro* against a wide range of human tumor cells." (Blick 2986, left col.)

Principles of Law

"[D]uring examination proceedings, claims are given their broadest reasonable interpretation consistent with the specification." *In re Hyatt,* 211 F.3d 1367, 1372 (Fed. Cir. 2000).

"[D]uring patent prosecution when claims can be amended, ambiguities should be recognized, scope and breadth of language explored, and clarification imposed." *In re Zletz*, 893 F.2d 319, 322 (Fed. Cir. 1989).

Analysis

Scannon discloses an antibody (XMMME-001) that binds to an antigen on melanoma cells and delivers a cytotoxic compound to those cells. Ferris discloses that immunoconjugates, like those of Scannon, can be made using any of a variety of cytotoxic compounds, including TNF. In view of these teachings, it would have been obvious to modify Scannon's immuno-conjugate to include TNF bound to the XMMME-001 antibody because Ferris teaches that TNF is a cytotoxic compound that is useful in immuno-conjugates.

Appellant argues that the Examiner has not established that Scannon's antibody binds to a *cell-surface* antigen, as required by the claims (Appeal

Br. 7-9). However, the Examiner has provided sufficient evidence to support a finding that XMMME-001 binds to an antigen on the cell surface: Oratz refers to Scannon's antibody, and Martin cites Oratz as showing that antibodies against the surface antigen HMWMAA have been used in anti-melanoma immunoconjugates. Ferrone also provides evidence that HMWMAA is a cell surface antigen, and Ashcroft provides evidence that HMWMAA has the same molecular weight (240 kD) as the antigen bound by XMMME-001. The Examiner's evidence supports a conclusion that XMMME-001 binds to HMWMAA, a cell-surface antigen.

Appellant also argues that the Examiner's reliance on the references discussed above "is unavailing to the Examiner because it requires reliance upon a reference from 2006," i.e., Ashcroft (Appeal Br. 8). This argument is not persuasive because post-filing evidence can be relied on to show the properties inherent in a prior art product. *See In re Wilson*, 311 F.2d 266, 268-69 (CCPA 1962). In any event, even if Ashcroft was omitted, Oratz, Martin, and Ferrone are adequate to support the Examiner's finding.

Appellant also argues that the claims require "*pre-testing* the patient" (Reply Br. 3) to determine that "cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site" (Appeal Br. 9) before administering the immunoconjugate.

We do not agree with Appellant's interpretation of the claims. Claim 26 requires that "it has been determined that cells of the patient's cancer express an antigen recognized and bound by" the immunoconjugate. The preamble of claim 26 recites a "method of treating cancer in a human patient." Claim 7, which depends from claim 26, states that "said cancer is selected from the group consisting of breast cancer, cervical carcinoma and

melanoma." Thus, read in the context of the claims as a whole, the "wherein" clause of claim 26 merely requires that the patient's type of cancer (e.g., breast cancer or melanoma) expresses an antigen that is bound by the antigen-recognizing protein in the immunoconjugate that is administered.

Appellant's claim interpretation also fails because the claimed method is defined by the three recited steps: identifying a patient with a tumor, obtaining an immunoconjugate composition, and administering the composition. Claim 26 does not recite a step of testing the patient's tumor cells to determine which tumor-associated antigen(s) they express. If Appellant intends such a step to be part of the claimed method, the claims should be amended to unambiguously recite it. *See Zletz*, 893 F.2d at 322 ("[D]uring patent prosecution when claims can be amended, ambiguities should be recognized . . . and clarification imposed.").

With respect to the rejection of claim 23, Appellant relies on the same arguments discussed above (Appeal Br. 11-12). With respect to claim 16, Appellant argues that "Blick does not relate to immunotoxins or similar targeted therapy at all. Thus, a person of skill in the art . . . would have very little reason to believe that whatever 'success' was reported therein could be repeated by a targeted TNF alpha conjugate." (*Id.* at 10.)

This argument is not persuasive. Ferris discloses that TNF is cytotoxic (FF 5). Blick discloses that there are two types of TNF, TNF-α and TNF-β, and both are cytotoxic against human tumor cells (FF 16). We agree with the Examiner that a person of ordinary skill in the art would have considered it obvious, based on Blick, to use TNF-α as the cytotoxic agent in the immuno-conjugate suggested by Scannon and Ferris.

Conclusion of Law

Appellant has not shown that the Examiner erred in finding that Scannon discloses an antibody that binds to a cell-surface antigen. The claims do not require confirming that a patient's tumor expresses the specific antigen that is recognized by the agent that is administered.

REJECTION BASED ON FRANKEL AND FERRIS

Issue

The Examiner has rejected claims 7, 24, and 26-30 under 35 U.S.C. § 103(a) as obvious in view of Frankel and Ferris (Ans. 10). The Examiner finds that Frankel teaches "a method of treating breast cancer in a human comprising administering an antibody-ricin A toxin conjugate wherein the antibody binds a breast cancer antigen" (Ans. 10). The Examiner concludes that it would have been obvious to "substitute TNF for the ricin A toxin of the antibody conjugate taught by Frankel et al because Ferris teach[es] that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody" (*id.* at 11).

Appellant contends that Frankel does not disclose a cell surface antigenic marker that is expressed more on tumor cells than on non-target cells, as recited in claim 26, because "[*n*]*one* of the antibodies presented in Frankel bind to *all* of the breast cancer cells [sic] lines or tissue sections tested in excess of that found at other non-cancerous sites" (Appeal Br. 5). Appellant concludes that "[t]herefore, there are breast cancer cell lines, tissues, and *tumors* that *do not express* antigens to Frankel's antibodies. At most, a given Frankel patient's cancer might express a given antigen" but that does not suffice to show inherency (*id.*).

The issue with respect to this rejection is: Has Appellant shown that the Examiner erred in concluding that Frankel discloses an antibody that binds an antigenic marker that is expressed at greater concentrations on tumor cells than on non-target cells?

Additional Findings of Fact

17. Frankel discloses immunotoxins comprising the ricin A chain toxin and "monoclonal antibodies that . . . bind selectively to human breast cancer cells" (Frankel, col. 1, ll. 48-51).

18. Frankel discloses that "[a]ntibodies were deemed to bind selectively to breast cancer if they bound strongly to less than about ⅓ of the normal tissues and blood cell types" (*id.* at col. 3, ll. 41-43).

19. Frankel discloses that "[s]ixteen of the antibodies exhibited acceptable immunotoxin activity . . . against at least one of [four] breast tumor lines" (*id.* at col. 3, ll. 51-54).

20. Frankel discloses that each of the sixteen antibodies tested bound to some breast cancer tissue sections (*id.* at col. 10, Table 2) and breast cancer cell lines (*id.* at col. 10-11, Table 3) but did not bind some or all types of normal tissue (*id.* at col. 7-10, Table 1).

21. Frankel discloses that "[t]here was no binding to platelets, red cells, lymphocytes, [or] monocytes. . . . None of the antibodies bound to fibroblasts." (*Id.* at col. 9-10, legend to Table 1).

Analysis

Appellant does not dispute the Examiner's conclusion that it would have been obvious to substitute Ferris' TNF for the ricin A chain toxin in Frankel's immunotoxin. Instead, Appellant contends that Frankel does not

teach an antigenic marker that is expressed in greater concentration on tumor cells than on normal cells, as required by the claims (Appeal Br. 5). Appellant points to the breast cancer labeled "R" in Frankel's Table 2, and reasons that the "only three antibodies that bind to R also bind to a significant amount of normal tissue types" (*id.*). Appellant concludes that R is an example of a "breast cancer [that] *does not express an antigen recognized by Frankel's antibodies* at higher concentrations than normal tissue" (*id.*).

As we understand it, Appellant's position is that the claims require administering an immunotoxin that binds to an antigen that is expressed at higher concentrations on the target tumor cells than on every type of non-target cells. This is not the broadest reasonable interpretation of the claim language.

Claim 26 recites identifying a patient with a tumor having "cells for targeting" that express "a cell surface antigenic marker at concentrations in excess of that found at other non-target sites." Under the broadest reasonable interpretation, this limitation does not require the antigenic marker to be expressed at lower levels on every other type of cell than on the target tumor cells, only that the antigenic marker is expressed at greater concentration on the target tumor cells than on *some* other, non-target cells.

Frankel discloses antibodies that bind to antigenic markers expressed on breast cancer cells (FF 20). Frankel also discloses that its antibodies bind strongly to less than ⅓ of normal tissues and blood cell types (FF 18), that each of the tested antibodies does not bind to at least some types of normal cells (FF 20), and that none of the tested antibodies binds to platelets, red cells, lymphocytes, monocytes, or fibroblasts (FF 21). Thus, Frankel

discloses antibodies that bind to antigenic markers that are expressed at greater concentrations on breast cancer cells than on at least some types of non-target cells. No more is required by the claim language.

Appellant also argues that the claims require "that a determination be made that the patient's cancer expresses the targeted antigen," which is not suggested by Frankel (Appeal Br. 6). This argument is not persuasive for the reasons discussed above with respect to the rejections based on Scannon and Ferris.

Conclusion of Law

Appellant has not shown that the Examiner erred in concluding that Frankel discloses an antibody that binds an antigenic marker that is expressed at greater concentrations on tumor cells than on non-target cells.

REJECTION BASED ON MATTES AND FERRIS

Issue

The Examiner has rejected claims 7, 24, 26-29, and 31 under 35 U.S.C. § 103(a) as obvious in view of Mattes and Ferris. The Examiner finds that Mattes teaches "a method for treating cervical carcinoma in a human comprising administering a monoclonal antibody, MH94, conjugated to a toxin to kill cancer cells" and that the MH94 antibody binds to an antigen that is expressed on cervical carcinoma cells at a greater concentration than on other cells (Answer 12). The Examiner concludes that it would have been obvious to "substitute TNF for the toxin of the antibody conjugate taught by Mattes et al because Ferris teach[es] that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody" (*id.* at 13).

Appellant contends that "[c]laim 31 is specifically directed to targeting cervical cancer and no reference in the record has been cited that correctly accounts for such a limitation" (Appeal Br. 12). Appellant contends that Mattes does not disclose an antigen specific to cervical cancer cells because MH94 "binds only weakly to cervical carcinoma cell lines, but . . . also *binds an excess of normal adult and fetal tissues*" (*id.* at 13)

The issue with respect to this rejection is: Has Appellant shown that the Examiner erred in concluding that Mattes discloses an antibody that binds an antigenic marker that is expressed at greater concentrations on cervical cancer cells than on non-target cells?

Additional Findings of Fact

22. Mattes discloses monoclonal antibody MH94 (Mattes, col. 7, l. 11).

23. Mattes discloses that MH94 binds to a cervical carcinoma cells (*id.* at col. 7-8, Table I).

24. Mattes discloses that the MH94 antigen was also "detected in the acinar and duct lining cells of the pancreas, the epithelial cells of the ureter, breast, pancreas, cervix and urinary bladder and the sweat and sebaceous glands of the skin" (*id.* at col. 11, l. 66 to col. 12, l. 1).

25. Mattes discloses that MH94 does not bind to normal fibroblasts, melanocytes, or kidney epithelia (*id.* at col. 9-10, Table I).

26. Mattes discloses that "[b]lood leukocytes were negative" for the MH94 antigen (*id.* at col. 11, ll. 64-65).

27. Mattes discloses that the five disclosed monoclonal antibodies, including MH94, "detect[ ] highly restricted antigens" (*id.* at col. 12, ll. 9-10).

28. Mattes discloses "the treatment of ovarian, cervical, or endometrial tumors in a patient wherein the monoclonal antibody recognizing the cell antigen . . . is administered to the patient in an amount effective to inhibit the growth or proliferation of cancer cells" (*id.* at col. 14, ll. 27-33).

29. Mattes discloses that "the antibody is tagged with a potentially tissue destructive agent which causes destruction of the cancer cells" (*id.* at col. 14, ll. 34-36).

Analysis

Appellant does not dispute the Examiner's conclusion that it would have been obvious to use Ferris' TNF as the tissue destructive agent in the immunotoxin disclosed by Mattes. Instead, Appellant contends that Mattes does not teach an antigenic marker that is expressed in greater concentration on cervical carcinoma cells than on normal cells, as required by claim 31 (Appeal Br. 12-13).

This argument is not persuasive for the reasons discussed above with respect to the rejection based on Frankel and Ferris. Claim 31 depends from claim 26 and therefore includes the same limitation of identifying a patient with a tumor having "cells for targeting" that express "a cell surface antigenic marker at concentrations in excess of that found at other non-target sites." As previously discussed, under the broadest reasonable interpretation, this limitation requires only that the antigenic marker is expressed at greater concentration on the target tumor cells than on some, not all, non-target cells.

Mattes discloses that the antigen bound by antibody MH94 is not expressed on normal fibroblasts, melanocytes, kidney epithelia, or blood leukocytes (FFs 25, 26). Thus, the MH94 antibody disclosed by Mattes binds

to an antigenic marker that is expressed at greater concentrations on cervical cancer cells than on at least some types of non-target cells, meeting the disputed limitation, when given its broadest reasonable interpretation.

Conclusion of Law

Appellant has not shown that the Examiner erred in concluding that Mattes discloses an antibody that binds an antigenic marker that is expressed at greater concentrations on cervical cancer cells than on non-target cells.

SUMMARY

We affirm the rejection of claim 26 under 35 U.S.C. § 103(a) based on Scannon and Ferris, with evidence provided by Kirkwood. Claims 7, 10, 13, 14, 21, 24, 25, 27-29, and 32 fall with claim 26 because they were not argued separately. 37 C.F.R. § 41.37(c)(1)(vii). We also affirm the rejection of claim 16 under 35 U.S.C. § 103(a) based on Scannon, Ferris, and Blick, and the rejection of claim 23 under 35 U.S.C. § 103(a) based on Scannon, Ferris, and Ghose.

We affirm the rejection of claim 26 under 35 U.S.C. § 103(a) based on Frankel and Ferris. Claims 7, 24, and 27-30 fall with claim 26 because they were not argued separately. 37 C.F.R. § 41.37(c)(1)(vii).

We affirm the rejection of claim 26 under 35 U.S.C. § 103(a) based on Mattes and Ferris. Claims 7, 24, 27-29, and 31 fall with claim 26 because they were not argued separately. 37 C.F.R. § 41.37(c)(1)(vii).

TIME PERIOD FOR RESPONSE

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a).

AFFIRMED

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